WEST Search History

DATE: Saturday, January 04, 2003

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=OR			
L7	(4507466)[pn]	1	L7
L6	dendrimer same (Cys or cysteine)	18	L6
L5	dendrimer near cysteine	0	L5
L4	dendrimer near alanine	0	L4
L3	dendrimer	878	L3
L2	e9 adj starburst	3	L2
L1	(4766106)[PN] OR (4847325)[PN]	2	L1

END OF SEARCH HISTORY

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     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 12:35:05 ON 04 JAN 2003
L1         11034 S WONG H?/AU OR RHODE P?/AU OR WEIDANZ J?/AU OR GRAMMER S?/AU O
L2             0 S L1 AND ((MULTIMER OR MULTIVALENT) AND MHC)
L3            14 S L1 AND (FUSION AND MHC)
L4            10 DUP REM L3 (4 DUPLICATES REMOVED)
L5            13 S (SOLUBLE (10N)  (CLASS (1N)  II) (10N) FUSION) AND MHC
L6            11 DUP REM L5 (2 DUPLICATES REMOVED)
L7            10 S L6 NOT L4
L8             0 S L5 AND DENDRIMER?
L9             7 S DENDRIMER? AND MHC
L10            3 DUP REM L9 (4 DUPLICATES REMOVED)
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=> s 3dt52.5
             6 3DT52
       1759720 5
L1            6 3DT52.5
                (3DT52(W)5)

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2            6 DUP REM L1 (0 DUPLICATES REMOVED)

=> dis l2 1-6 ibib abs

L2   ANSWER 1 OF 6     MEDLINE
ACCESSION NUMBER:   1998026161     MEDLINE
DOCUMENT NUMBER:    98026161   PubMed ID: 9379039
TITLE:              Rigidification of the alpha2 helix of an MHC class I
                    molecule by a valine to proline mutation in position 165
                    does not prevent peptide-specific antigen presentation.
AUTHOR:             Plaksin D; Polakova K; Mage M G; Margulies D H
CORPORATE SOURCE:   Laboratory of Immunology, National Institute of Allergy and
                    Infectious Diseases, National Institutes of Health,
                    Bethesda, MD 20892-1892, USA.
SOURCE:             JOURNAL OF IMMUNOLOGY, (1997 Nov 1) 159 (9) 4408-14.
                    Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:        199711
ENTRY DATE:         Entered STN: 19971224
                    Last Updated on STN: 19971224
                    Entered Medline: 19971112
AB   Although classical MHC class I glycoproteins bind peptide Ags for display
     at the cell surface, some MHC class I-related molecules such as the
     neonatal Fc receptor (FcRn) execute their function without binding peptide
     ligands. The three-dimensional structure of the FcRn suggested that a
     substitution of the conserved valine at position 165 of the alpha2 helix
     by proline contributed to a kink in the position of this helix relative to
     the alpha1 helix, and resulted in closing of the potential peptide-binding
     cleft. To test the contribution of proline 165 to the occlusion of the
     cleft and the binding of potential antigenic peptides, we introduced this
     mutation into the classical murine MHC class I molecule, H-2Dd, and
     characterized the ability of such a mutant to present peptide Ags to
     either a peptide-specific, H-2Dd-restricted T cell hybridoma (B4.2.3), or
     an allospecific, peptide-dependent, T cell hybridoma (3DT52.
     5.8). We show that the V165P mutation, expressed at the cell
     surface either in H-2Dd or in a single chain membrane version of H-2Dd,
     fails to eliminate recognition of the peptide/MHC complexes by two
     different T cells. Evaluation of a panel of synthetic substituted peptides
     suggests that subtle differences in the fine specificity of presentation
     can be discerned. Thus, the proline substitution at position 165 of FcRn
     and some other class I-like molecules is not the sole cause of the lack of
     peptide presentation.

L2   ANSWER 2 OF 6     MEDLINE
ACCESSION NUMBER:   96173773     MEDLINE
DOCUMENT NUMBER:    96173773   PubMed ID: 8596036
TITLE:              Mutations in human CD4 impair the functional interaction
                    with different human and mouse class II isotypes and
                    alleles.
AUTHOR:             Fleury S; Huang B; Zerbib A; Croteau G; Long E O; Sekaly R
                    P
CORPORATE SOURCE:   Immunology Laboratory, Montreal Clinical Research
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Institute, Canada.
SOURCE: JOURNAL OF IMMUNOLOGY, (1996 Mar 1) 156 (5) 1848-55.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199604
ENTRY DATE: Entered STN: 19960424
Last Updated on STN: 19960424
Entered Medline: 19960415

AB The structure-function of the CD4-class II MHC interaction was investigated. Two functional assays were used to assess the responses of the **3DT52.5**.8 murine T cell hybridoma expressing human CD4 (h-CD4) or murine CD4 (m-CD4). First, we determined the responses of the CD4+ and CD4-effector cells toward DAP-3 cells co-expressing the cognate alloantigen H-2Dd together with several human (DRw52b, DR4-Dw4, DR2A, and DPw2) and murine (I-Ab, I-Ak, IA alpha b I-A beta k and I-Ek) class II alleles and isotypes. We found that h-CD4 and m-CD4 strongly enhance the T cell response to H-2Dd, demonstrating that interspecies CD4/class II interactions occur efficiently. Furthermore, mutations in h-CD4 at positions 19, 89, and 165 markedly reduced the interaction with both human class II and mouse class II, indicating that the structural features of this cross-species interaction are strongly conserved. This was further supported by the finding that a h-CD4 deletion mutant (deletion F43-S49) interacted with both human and murine class II. Moreover, as 3DT cells express the responsive V beta element for the bacterial superantigen staphylococcal enterotoxin B, a co-receptor assay was conducted. DAP-3 cells expressing only class II molecules were used as APCs to present staphylococcal enterotoxin B to h-CD4+ and m-CD4+ T cells. h-CD4 and m-CD4 were able to enhance the T cell response to staphylococcal enterotoxin B, further demonstrating the conservation of the CD4-class II MHC interaction.

L2 ANSWER 3 OF 6 MEDLINE
ACCESSION NUMBER: 87215948 MEDLINE
DOCUMENT NUMBER: 87215948 PubMed ID: 3034434
TITLE: The role of the L3T4 molecule in mitogen and antigen-activated signal transduction.
AUTHOR: Rosoff P M; Burakoff S J; Greenstein J L
CONTRACT NUMBER: AM07715 (NIADDK)
AM31405 (NIADDK)
DK34605 (NIDDK)
SOURCE: CELL, (1987 Jun 19) 49 (6) 845-53.
Journal code: 0413066. ISSN: 0092-8674.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198707
ENTRY DATE: Entered STN: 19900303
Last Updated on STN: 19970203
Entered Medline: 19870723

AB We investigated the role of the L3T4 molecule in mitogen and antigen-initiated signal transduction in the L3T4(+) murine T cell hybridoma, **3DT52.5**.9 and an L3T4(-) variant, **3DT52.5**.24. Both Concanavalin A (Con A) and specific antigen stimulated increases in cytosolic-free calcium ([Ca2+]i), phosphatidylinositol turnover, and interleukin-2 (IL-2) production in both cell lines. About 85% of the stimulated rise in [Ca2+]i was from an extracellular source. Anti-L3T4 monoclonal antibody (MAb) inhibited 90% of antigen- and 50% of Con A-stimulated increases in [Ca2+]i and IL-2 production but had no effect on the ability of either activation signal to stimulate phosphatidylinositol turnover in the parent L3T4(+) cells.

Stimulus-response coupling in the L3T4(-) cells was unaffected by the MAb. The anti-L3T4-insensitive increase in [Ca2+]i induced by Con A was inhibited by EGTA, suggesting that this mitogen also stimulated an influx of Ca2+ via an additional transport mechanism distinct from that stimulated by antigen. The fact that anti-L3T4 antibodies inhibit antigen and Con A-stimulated Ca2+ transport and IL-2 production without affecting phosphatidylinositol turnover suggests that L3T4 may play a critical role in modulating the activation of the T cell receptor-associated Ca2+ transporter in T cell stimulus-response coupling.

L2 ANSWER 4 OF 6 MEDLINE
ACCESSION NUMBER: 85236163 MEDLINE
DOCUMENT NUMBER: 85236163 PubMed ID: 3925069
TITLE: Role of L3T4 in antigen-driven activation of a class I-specific T cell hybridoma.
AUTHOR: Greenstein J L; Malissen B; Burakoff S J
CONTRACT NUMBER: AI-17258 (NIAID)
CA-07324 (NCI)
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1985 Jul 1) 162 (1) 369-74.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198508
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19980206
Entered Medline: 19850815

AB The expression of L3T4/Lyt-2 on murine T cells has led to the association of these surface markers with recognition of either class II or class I major histo-compatibility complex (MHC) antigens. It has been suggested that these T cell surface antigens interact with nonpolymorphic determinants on MHC antigens. We have examined the role of L3T4 in the recognition of H-2Dd by the T cell hybridoma, **3DT52.5**. Mouse L cells transfected with either the H-2Dd gene, or with both the alpha and beta genes of I-Ak and the H-2Dd gene have been used to assess the role of an L3T4/1a interaction at varying doses of H-2Dd. A role of L3T4 in activation of **3DT52.5** becomes evident only at limiting doses of antigen. It appears that an L3T4/1a interaction can influence T cell function during suboptimal stimulation, implying that the L3T4/1a interaction serves to raise the functional affinity of interaction between the T cell and the antigen-bearing cell.

L2 ANSWER 5 OF 6 MEDLINE
ACCESSION NUMBER: 84163773 MEDLINE
DOCUMENT NUMBER: 84163773 PubMed ID: 6200564
TITLE: The role of L3T4 in recognition of Ia by a cytotoxic, H-2Dd-specific T cell hybridoma.
AUTHOR: Greenstein J L; Kappler J; Marrack P; Burakoff S J
CONTRACT NUMBER: AI-17258 (NIAID)
AI-18785 (NIAID)
CA-07324 (NCI)
SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1984 Apr 1) 159 (4) 1213-24.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198405
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19980206
Entered Medline: 19840514

AB The expression of T4/T8 surface markers on human T cells and of L3T4/Lyt-2 on murine T cells has lead to the association of these surface markers with recognition of either class II or class I major histocompatibility complex (MHC) antigens. It has been suggested that these T cell surface antigens interact with MHC antigens. We have examined the role of L3T4 in the recognition of Dd by the T cell hybridoma, **3DT52.5** . This T cell hybridoma was found to be specific for the N/Cl domain of Dd. The recognition of a class I antigen by an Lyt-2-, L3T4+ T cell hybridoma allowed the separate evaluation of interactions between L3T4/Ia and the T cell antigen receptor, Dd. Recognition by this hybridoma resulted in the production of interleukin 2 (IL-2) and cytolytic activity. Antibody blocking experiments have demonstrated that L3T4 was involved in triggering the effector function of **3DT52.5** only on Ia+ stimulator or target cells. We have demonstrated that an L3T4+, Dd-specific T cell hybridoma, **3DT52.5**, uses the L3T4 molecule to directly interact with nonpolymorphic Ia determinants.

L2 ANSWER 6 OF 6 MEDLINE
ACCESSION NUMBER: 84009070 MEDLINE
DOCUMENT NUMBER: 84009070 PubMed ID: 6604749
TITLE: An IL 2-secreting T cell hybridoma that responds to a self class I histocompatibility antigen in the H-2D region.
AUTHOR: Endres R O; Marrack P; Kappler J W
CONTRACT NUMBER: AI-18785 (NIAID)
SOURCE: JOURNAL OF IMMUNOLOGY, (1983 Oct) 131 (4) 1656-62. Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198311
ENTRY DATE: Entered STN: 19900319
Last Updated on STN: 19970203
Entered Medline: 19831123

AB A self-reactive T cell hybridoma that secretes IL-2 in response to H-2d haplotype cells resulted from a fusion of BALB/cBy lymph node cells with the AKR thymoma BW5147. The lymph node cells used had been enriched for cells reactive to (TG)-A--L, but neither this antigen nor fetal calf serum were required for stimulation of the hybridoma designated **3DT52.5**. The gene product responsible for stimulation mapped to the H-2D region. Allogeneic cells of the b, f, k, q, and s haplotypes failed to stimulate. Not all H-2d haplotype cells were effective stimulators of **3DT52.5**. Peritoneal cells and splenic B cells were much more stimulatory than splenic T cells. Most tumor cell lines of H-2d derivation and of B cell or macrophage/monocyte lineage were stimulatory, whereas H-2d T cell lines were not. The capacity to stimulate **3DT52.5** did not correlate with the ability to stimulate I region-restricted hybridomas, or with the ability to be induced to stimulate such hybridomas. Stimulatory cell lines did not apparently produce a soluble factor required for stimulation, and negative cell lines were not inhibitory. The monoclonal antibody 27-11-13, which reacts with H-2D of the b, d, and q haplotypes, inhibited stimulation of **3DT52.5** but did not inhibit stimulation of the sibling hybridoma 3DT18.11, which responds to (TG)-A--L plus I-Ad. Conversely, the monoclonal anti-I-Ad antibody MK-D6 inhibited stimulation of 3DT18.11 but not **3DT52.5**. Although it is clear that **3DT52.5** recognizes a class I antigen coded for in the H-2D region, the precise molecular nature of the antigen is unknown. The structure of the antigen receptor on this hybridoma may prove to be of interest when it can be compared with receptors found on T cell hybridomas restricted by class II histocompatibility antigens.